Public Health

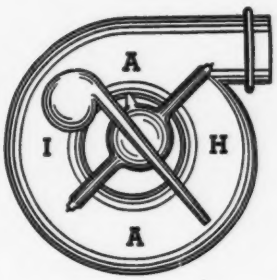
MAR 15 1950

AMERICAN

INDUSTRIAL HYGIENE

ASSOCIATION

QUARTERLY



VOLUME 10 JUNE, 1949 NUMBER 2

AMERICAN INDUSTRIAL HYGIENE ASSOCIATION

Officers

President
E. C. BARNES — Westinghouse Electric Corporation

President-Elect
ALLEN D. BRANDT, Sc.D. — Bethlehem Steel Company

Past President
JAMES H. STERNER, M.D. — Eastman Kodak Company

Secretary
W. G. HAZARD — Owens-Illinois Glass Company

Treasurer
C. H. MEHAFFEY — Mine Safety Appliances Company

Executive Secretary
HENRY F. SMYTH, JR., Ph.D. — Mellon Institute, 4400 Fifth Avenue, Pittsburgh 13, Pennsylvania

Board of Directors

ANNA M. BAETJER, Sc.D.
The Johns Hopkins School of Hygiene and Public Health

CARLTON E. BROWN, Sc.D.
Camp Dedrick

ROBERT M. BROWN
Yale University Institute of Occupational Medicine and Hygiene

A. G. CRANCH, M.D.
Union Carbide and Carbon Corporation

THEODORE F. HATCH
Industrial Hygiene Foundation of America

WILLIAM E. MCCORMICK
B. F. Goodrich Company

L. M. PETRIE, M.D.
Georgia Department of Public Health

BERNERD D. TEBBENS, Sc.D.
University of California

HERBERT T. WALWORTH
Lumbermens Mutual Casualty Company

CHARLES R. WILLIAMS, Ph.D.
Liberty Mutual Insurance Company

N. V. HENDRICKS
Standard Oil Company (New Jersey)

Editorial Committee

F. S. MALLETTE, Chairman — Firestone Tire & Rubber Company
PHILIP DRINKER — Harvard School of Public Health
LEON LEWIS, M.D. — University of California
H. F. SMYTH, JR. — Mellon Institute
F. A. VAN ATTA — National Safety Council

American Industrial Hygiene Association Quarterly

WARREN A. COOK, Editor — Zurich Insurance Companies
HERBERT J. WEBER, Associate Editor — American Brake Shoe Company
VINCENT J. CASTROP, Advertising Editor — General Motors Corporation

AMERICAN
INDUSTRIAL HYGIENE
ASSOCIATION
QUARTERLY

Volume 10 JUNE, 1949 Number 2

This Issue

A STUDY OF MERCURY EXPOSURE
M. O. SHOIB, M.D., LEONARD J. GOLDWATER, M.D. and MARTIN SASS 29

FURTHER OBSERVATIONS ON CONTROL OF A LEAD HAZARD IN THE BRASS FOUNDRY INDUSTRY
L. E. HAMLIN, M.D. and H. J. WEBER 32

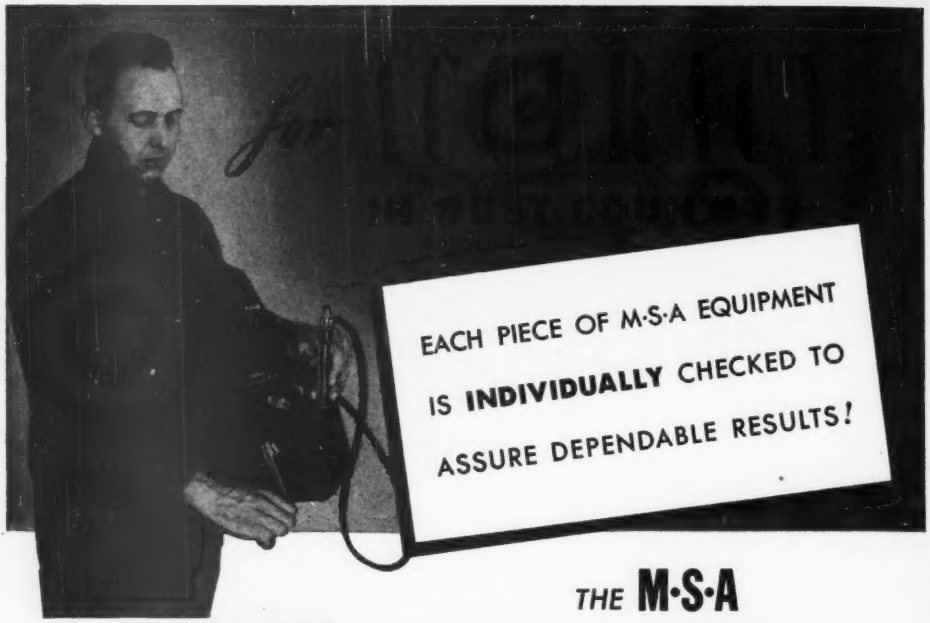
AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS
Eleventh Annual Meeting
AMERICAN INDUSTRIAL HYGIENE ASSOCIATION
Tenth Annual Meeting
E. D. SALLEE and C. O. SAPPINGTON, M.D., D.P.H. 37

THE USE OF BERYLLIUM IN NEW YORK STATE
JOHN E. SILSON, M.D., LAWRENCE P. BENJAMIN, CH. E. and SAMUEL C. WILSON 45

AMERICAN INDUSTRIAL HYGIENE ASSOCIATION QUARTERLY, an Official Publication of the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION, published quarterly (March, June, September, December) by INDUSTRIAL MEDICINE PUBLISHING COMPANY, Chicago (publishers also of INDUSTRIAL MEDICINE and SURGERY, issued monthly). STEPHEN G. HALOS, President; A. D. CLOUD, Publisher; DORIS FLOURNOY, Editorial Assistant; HAROLD A. HOOPER, Treasurer; STEPHEN G. HALOS, Advertising and Business Manager. PUBLICATION, EDITORIAL and EXECUTIVE Offices, 605 North Michigan Avenue, Chicago 11, Illinois. Subscription $2.00 per year in the United States; $2.50 per year in Canada; $3.00 per year in other countries. Single copies 75 cents. Copyright, 1949, the American Industrial Hygiene Association.

Entered as second class matter May 3, 1948 at the post office at Sheboygan, Wis., under the Act of March 3, 1879.

UNIVERSITY OF MICHIGAN LIBRARIES



THE **M·S·A**

MIDGET IMPINGER

AND AUXILIARY EQUIPMENT

New all-glass impinger tube (as illustrated) and standard rubber-stoppered tubes—both carefully checked for correct flow.

The widely-employed M.S.A. Midget Impinger is invaluable in survey work because of its precisely-checked components, which enable reproducible results. Each piece is individually examined and calibrated before assembly by actual flow meter tests in which all nozzles are limited to a variance of not more than 4% from the required flow of 1/10 cu. ft. per minute at 12" of water suction. Every factor is made constant by M.S.A. laboratory control, assuring the technician of *equipment dependability* in his measurement of hazards.

Above: M·S·A Dust Counting Cell, checked for 1mm depth. Left: M·S·A Dust Counting Microscope.

M·S·A Dust Counting Microscope

Specially designed for dust counting, this quality instrument has a draw tube set and locked, after laboratory check, at exactly 100x magnification.

MINE SAFETY APPLIANCES COMPANY

BRADDOCK, THOMAS AND MEADE STREETS · PITTSBURGH 8, PA.

At Your Service:

54 BRANCH OFFICES IN THE UNITED STATES AND CANADA

AMERICAN
INDUSTRIAL HYGIENE
ASSOCIATION
QUARTERLY

Volume 10 JUNE, 1949 Number 2

A Study of Mercury Exposure

M. O. SHOIB, M.D., LEONARD J. GOLDWATER, M.D., MARTIN SASS, B.S.
School of Public Health, Columbia University

ALTHOUGH MERCURY has been recognized as an industrial poison for several hundred years there are still many features of mercury poisoning which are not well understood. The present report is presented as a small contribution to the available information on the subject of chronic industrial mercury exposures and because it calls attention to possible hematologic changes which have not attracted great attention.

The plant in which the studies were conducted is engaged in the manufacture of several mercury compounds, principally the bichlorides, the oxides and phenyl mercuric acetate. The manufacturing processes are not enclosed so that there is considerable manual operation. Ventilation is provided mainly by windows and doors supplemented by a few inefficient exhaust hoods.

Procedures

SAMPLING of air was done with the midget impinger at the rate of five liters per minute for 20 minutes using aqueous potassium iodide as the absorbing medium. A total of seven samples in duplicate were analyzed by Barnes'[1] method. Additional simultaneous sampling was done with the General Electric mercury vapor analyzer.

Morning urine samples, collected before the employees began work, were obtained from 18 employees. These were analyzed quantitatively for mercury by a modification of the method of Gettler and Lehman.[2] Casual urine samples are examined for albumin by the plant physician about every three months. This procedure was not repeated in the present survey as the periodic examination had been done quite recently. Hematologic studies were performed on the 18 individuals who submitted urine samples for mercury analysis. Periodic physical examinations with particular emphasis on the detection of tremors and gingivitis are performed regularly by the plant physician. None of the men included in the present study exhibited any clinical signs or symptoms of mercury intoxication.

Results

THE CONCENTRATIONS of mercury found in the air samples which were collected with the midget impinger are given in Table I. It is apparent from Table I that all air samples contained mercury in excess of the generally accepted maximal allowable concentration of 0.1 mg. per cubic meter. Readings on the mercury vapor analyzer varied between 0.04 mg. and 0.85 mg. per cubic meter of air. The discrepancy between these findings and those on the impinger samples is believed to be due primarily to the insensitivity of the instrument to mercury-containing dust and to the interference

TABLE I.
ATMOSPHERIC MERCURY VALUES

Sample No.	Location or operation	Mg. Hg per cu. m. air
1*	Chlorinating chamber	0.31
2*	Removing oxide from rats	0.21
3	Mercury redistilling room	0.15
4	Shipping room	0.21
5	Control laboratory	0.34
6*	Chlorinators	0.44
7*	Oxide drying bags	0.35

* Sample taken at breathing zone of operator

Presented before the Metropolitan New York Section of the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION May 6, 1949.

of other chemicals which were used in the plant.

The urinary excretion of mercury is shown in Table II. The division line separates the series using the median urinary mercury values as the dividing point.

TABLE II.
URINARY MERCURY VALUES

Case No.	Time Employed	Mg. Hg per liter of urine
1	2 years	3.79
2	3 years	3.77
3	3.5 years	3.49
4	1 year	3.15
5	3 months	2.73
6	3 months	2.50
7	2 months	2.21
8	1 year	2.12
9	2 months	2.0
10	3.5 years	1.79
11	3.5 years	1.70
12	3.5 years	1.61
13	3.5 years	1.47
14	3.5 years	1.30
15	3.5 years	1.16
16	3.5 years	1.14
17	3.5 years	0.91
18	3 years	0.13

Since none of the men confined their activities to a single department or operation it is not possible to draw any correlation between atmospheric and urinary mercury concentrations. That such a correlation exists has recently been pointed out by Smith and Moskowitz.[3] Furthermore, no relationship between urinary excretion of mercury and clinical evidence of poisoning could be demonstrated since none of the employees exhibited signs or symptoms of mercurialism, even though urinary mercury levels above 3.0 mg. per liter were found in several cases. On the other hand, there appeared to be a relationship between urinary excretion and duration of exposure.

If the series is divided evenly, using the median urinary mercury value as the dividing point, it is found that all individuals in the low excretion group had been exposed for more than three years while 66% of the high excretion group had been exposed for one year or less. This phenomenon of diminishing urinary mercury excretion after prolonged exposure has been demonstrated experimentally by Ashe, Largent and Hubbard.[4] Possible mechanisms to explain the declining urinary excretion may be (a) failure of the excretory mechanism and increased body storage; (b) establishment of other routes of excretion, e.g. bile, sweat, etc.; (c) diminished mercury absorption through the lungs.

Hematologic Observations

RELATIVELY little has been written concerning blood changes in chronic industrial mercury intoxication. Occasional references are found to "relative lymphocytosis" but this appears to be nothing more than a reflection of the acceptance of misleading normal standards. The occurrence of "mild secondary anemia" among workers exposed to mercury compounds is also mentioned[5] but studies reported by Goldwater[6] tend to indicate that high rather than low hemoglobin values are the rule.

Tables III to VIII contain the blood findings which were included in the present study and a comparison of these with a control group of male employees in a non-hazardous industrial operation.[7] The statistical comparisons given in Table IX show the following significant differences between the exposed and control groups: (a) hemoglobin values higher in the exposed group; (b) red cell count lower in the exposed group; (c) mean corpuscular volume (average size of red cells) larger in the exposed group; (d) absolute number of monocytes higher in the exposed group. Differences in total leucocytes and lymphocytes were not significant.

TABLE III.
DISTRIBUTION OF HEMOGLOBIN VALUES FOR CONTROL AND MERCURY GROUPS

Hb. (Gm./100 cc bld)	%		No. of Cases	
	Control	Mercury	Control	Mercury
< 11	0	0	0	0
11.0—11.0	0	0	0	0
12.0—12.9	2.4	0	2.0	0
13.0—13.9	30.9	0	25	0
14.0—14.9	49.5	27.8	40	5
15.0—15.9	14.8	61.1	12	11
16.0—16.9	2.4	11.1	2	2
Total	100%	100%	81	18

TABLE IV.
DISTRIBUTION OF ERYTHROCYTE COUNTS IN CONTROL AND MERCURY GROUPS

R. B. C. in millions per cu. mm.	%		No. of Cases	
	Control	Mercury	Control	Mercury
3.5—3.99	0	0	0	0
4.0—4.49	2.4	27.8	2	5
4.5—4.99	19.8	61.1	16	11
5.0—5.49	40.8	11.1	33	2
5.5—5.99	34.6	0	28	0
6 —6.49	2.4	0	2	0
Total	100%	100%	81	18

TABLE V.
DISTRIBUTION OF MEAN CORPUSCULAR VOLUME IN CONTROL AND MERCURY GROUPS

M.C.V. in	%		No. of Cases	
cu. microns	Control	Mercury	Control	Mercury
70—74	9.3	0	7	0
75—79	12.0	0	9	0
80—84	25.4	0	19	0
85—89	26.7	0	20	0
90—94	17.3	5.6	13	1
95—99	4.0	27.8	3	5
100—104	5.3	50.0	4	9
105—109	0	16.6	0	3
110—114	0	0	0	0
115—120	0	0	0	0
Total	100%	100%	75	18

TABLE VI.
DISTRIBUTION OF LEUCOCYTE COUNTS IN CONTROL AND MERCURY GROUPS

Leucocyte Count	%		No. of Cases	
(1000/cu. mm.	Control	Mercury	Control	Mercury
5000—5999	8.6	33.3	7	6
6000—6999	23.4	16.6	19	3
7000—7999	19.8	11.1	16	2
8000—8999	13.6	5.6	11	1
9000—9999	12.4	5.6	10	1
10000—10999	16.1	11.1	13	2
11000—11999	3.7	5.6	3	1
12000—12999	1.2	0	1	0
13000—13999	0	5.6	0	1
14000—14999	1.2	5.6	1	1
15000—15999	0	0	0	0
16000—16999	0	0	0	0
Total	100%	100%	81	18

TABLE VII.
DISTRIBUTION OF ABSOLUTE LYMPHOCYTE COUNTS IN CONTROL AND MERCURY GROUP

Lymphocytes	%		No. of Cases	
(absolute)	Control	Mercury	Control	Mercury
750—1000	0	0	0	0
1000—1999	11.1	38.9	9	7
2000—2999	42.0	33.3	34	6
3000—3999	24.8	11.1	20	2
4000—4999	17.2	0	14	0
5000—5999	3.7	11.1	3	2
6000—6999	1.2	5.6	1	1
Total	100%	100%	81	18

TABLE VIII.
DISTRIBUTION OF ABSOLUTE MONOCYTE VALUES IN CONTROL AND MERCURY GROUPS

Monocytes	%		No. of Cases	
(absolute)	Control	Mercury	Control	Mercury
200—299	23.4	11.1	19	2
300—399	16.2	16.6	13	3
400—499	24.8	11.1	20	2
500—599	12.4	5.6	10	1
600—699	8.7	11.1	7	2
700—799	4.8	11.1	4	2
800—899	4.8	5.6	4	1
900—999	3.7	11.1	3	2
1000—1099	1.2	11.1	1	2
1100—1199	0	5.6	0	1
Total	100%	100%	81	18

TABLE IX.
CONTROL AND MERCURY GROUPS

	R.B.C. (mil-lions)	Hb.	M.C.V.	W.B.C.	Lympho-cytes	Mono-cytes
Average of Group having more than 1.8 mg/1 of urine	15	4.6	104.2	7,200	2,600	640
Average of Group having less than 1.8 mg/1 of urine	15.5	4.8	98.2	8,900	3,100	770
Average of whole Group exposed	15.3	4.7	101.2	8,100	2,800	720
Average of Control Group	14.2	5.36	86.2	8,400	3,140	464
*Difference in Means	1	0.66	15.2	1.42	3.7	17.8
S.D. of Difference in Means	0.22	0.12	0.85	5.47	3	5.9
Difference						
S.D. of Diff.	4.5	5.5	17.8	0.25	1.25	3

* The mean values are calculated from frequency distribution and hence vary slightly from the average given.

Discussion and Conclusions

ALTHOUGH THE number of exposed individuals included in this study was quite small it was felt that the observations should be published since they include fairly complete environmental, clinical and laboratory data. Obviously it would be hazardous to draw any definite conclusions from this study but several points are suggested: 1. There may be no correlation between urinary mercury excretion and the appearance of clinical evidence of mercury poisoning. 2. On prolonged exposure to mercury, the urinary mercury excretion may diminish. 3. Chronic industrial exposure to mercury may result in increased hemoglobin values, lowered red blood cell counts, increased red cell size and absolute monocytosis.

References

BARNES, E. C.: Determining the Concentration of Mercury in Air. *A.I.H.A. Quarterly*, 7:26, June, 1946.

2. GETTLER, A. O. and LEHMAN, R. A.: A Simplified Procedure for the Determination of Mercury in Urine. *Am. J. of Clinical Pathology*, 8:161, September, 1938.

3. SMITH, A. R. and MOSKOWITZ, S.: Urinary Excretion of Mercury. New York State Department of Labor, *Monthly Labor Review*, 27:45, December 1948.

4. ASHE, W. F., LARGENT, E. J. and HUBBARD, D.: The Behavior of Mercury in the Animal Organism Following Inhalation of the Vapor of Metallic Mercury at Toxic Levels in Air. AMERICAN INDUSTRIAL HYGIENE ASSOCIATION meeting, Detroit, April 7, 1949.

5. A study of chronic Mercurialism in the Hatters Fur Cutting Industry. United States Public Health Service, *Public Health Bulletin*, No. 234, May, 1937.

6. GOLDWATER, L. J.: Physiology of the Bone Marrow in Relation to Industrial Intoxication. *Occupational Medicine*, 4:435, December, 1947.

7. GOLDWATER, L. J.: Disturbances in the Blood Following Exposure to Benzol. *J. of Laboratory and Clinical Medicine*, 24:957, March, 1941.

Further Observations on Control of a Lead Hazard in the Brass Foundry Industry

L. E. HAMLIN, M.D., Medical Director
H. J. WEBER, Industrial Hygiene Engineer
Medical Department
American Brake Shoe Company
Chicago, Illinois

OUR EXPERIENCE in developing a program for the control of exposure to lead, as outlined in a previous paper,[1] indicates that a majority of men in our plants have worked for many years in atmospheric concentrations well in excess of 1.5 milligrams of lead per 10 cubic meters of air without developing lead intoxication. From a study of over 15,000 spot samples of urine and 1,000 blood analyses for lead, we have evolved a series of urinary excretion values which we feel represent safe working conditions in our plants. We like to think of these adopted indices as having undergone "the road test" in their application to actual working environments.

When industrial exposure to lead is considered, one recognizes that many intangible factors are involved. Therefore the temptation to use any single test as an absolute yardstick in diagnosis should be resisted in favor of proper evaluation of *all* available significant clinical and hygienic data. For example, we have seen instances where a diagnosis of lead intoxication has been attempted on the basis of a single stipple cell count. The futility of making an interpretation on such a basis is obvious. We recognize too that research of other workers in the field has shown that hypertension, which is reflected first in the diastolic pressure, has developed in individuals with an exposure to lead of 20 years or more. It is realized that similar clinical findings may be anticipated in a number of the cases under consideration here but it is felt that the results of this five year study are of sufficient interest to warrant their publication at the present time.

It should be noted that prior to the application of present methods, no hygienic measures were in effect in these plants for controlling atmospheric contamination by lead. It is probable that a number of cases of plumbism went unrecognized or that others were erroneously ascribed to the effects of lead but the fact remains that even with the lack of control these plants operated on a full scale of productivity, without too much embarrassment because of lead intoxication. Since it may logically be assumed that the exposure was greater in this era, there would seem to be some indirect support for the observation that in actual practice many men handled atmospheric concentrations considerably greater than 1.5 milligrams of lead per 10 cubic meters of air without undue difficulty.

Controlled Conditions

TO CHECK the validity of these observations, the present study was initiated. In the first years of the investigation cases were not subjected to the same rigidly controlled conditions as in the last two years. In this latter period, clinical diagnosis has been more accurate and urine sampling more dependable. The advent of industrial nurses in most of the plants involved, has been of great value in developing and maintaining a more consistent procedure. Corrections for specific gravity have been done routinely for a year and a half but their validity has not been accurately determined thus far. Renal function tests have offered possible explanations in a few cases where discrepancies existed between concentration of lead in the blood and in the urine.

In the present study, excretion of urinary lead in approximately 1,000 men was observed over a period of five years. During this time it was apparent that lead intoxication rarely occurred unless the concentration in the spot sample was at least 300 micrograms of lead per liter of urine. Furthermore, in the majority of cases, intoxication did not develop unless this amount of excretion was maintained for a period of several weeks. In addition, many men with

Presented at the Local Section Meeting of the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION in Philadelphia, May 10, 1949.

values of from 400 to 1200 micrograms of lead per liter of urine showed no demonstrable plumbism. It is interesting to note that the experience of other groups using similar methods coincides with our own.

Since these findings were at variance with the present concept of dangerous levels of lead excretion, contamination of the samples was suspected. Repeated tests, including those concerned with possible laboratory sources of error and handling of specimens disproved this theory. Based on the foregoing, the following ranges of excretion of urinary lead were adopted for purposes of control. All urine and blood samples were analyzed polarographically according to previously published methods.[2]

TABLE I
RANGES OF EXCRETION OF LEAD

Range of Micrograms Per Liter Urine	Interpretation
0 to 199	No definite significance
200 to 299	Abnormal absorption (caution-range)
Over 300	Abnormal absorption (danger-range)

In order to test the usefulness of these ranges, representative spot samples of urine were selected from those individuals who submitted a significant number of specimens. These samples were used to determine if the concentration of excreted lead could be correlated with a diagnosis of plumbism as made by plant physicians. The group included 406 employees and analytical values have been determined from spot samples of urine collected without reference to the hour or to specific gravity.

The men were sampled approximately every three months during the first two years, monthly during the third year, and weekly during the last two years of the study. The majority were exposed to high concentrations of atmospheric lead for many years prior to the organization of a control program but no record of excreted lead exists on them.

For the purpose of this investigation, it must be assumed that the physician's diagnosis was reasonably accurate. Because statistically, he may have been wrong at many times in a positive as in a negative sense, diagnostic errors can offset each other. At any rate, they are not necessarily cumulative and, hence, do not invalidate the findings of the study.

Classifications

RANGES OF lead excretion were arbitrarily established for the purpose of classification, as follows:

Group 1 0—99 micrograms per liter of urine
Group 2 100—199 micrograms per liter of urine
Group 3 200—299 micrograms per liter of urine
Group 4 Over 300 micrograms per liter of urine

During the five year period the amounts of lead excretion were averaged on each man, following which he was placed in one of the ranges given above.

If intoxication occurred, quantities of excretion prior to the episode were averaged, as subsequent values had no bearing on the attack. The subject was then placed in the appropriate group according to his range, as shown in Tables II and III.

TABLE II
CASES SHOWING NO INTOXICATION—STUDY OF 406 MEN

	Distribution by Ranges		
Range Micrograms Per Liter Over 5 Year Period	Number of Men	% of Total	Average Number of Years Exposed
0—90	21	5.2	1.9
100—199	146	36.0	10.2
200—299	112	27.8	7.3
300 and over	63	15.5	7.2
TOTAL	342	84.2	8.4 Av.

NOTE: If more ranges were taken, it is evident that the distribution would approximate the normal curve of probability.

TABLE III
CASES SHOWING INTOXICATION—STUDY OF 406 MEN

	Distribution by Ranges		
Range Micrograms Per Liter Over 5 Year Period	Number of Men	% of Total	Average Number of Years Exposed
0—99	3	0.7	12.3
100—199	12	2.9	0.6
200—299	7	1.7	3.4
300 and over	42	10.3	11.7
TOTAL	64	15.7	9.2 Av.

The tables indicate that 342 or 84.2% of the 406 men in the study did not develop lead intoxication after an average exposure of 8.4 years regardless of concentrations of excreted lead. They also demonstrate that 64 men or 15.7% did develop intoxication after periods of exposure averaging 9.2 years.

A question might be asked: "Is an incidence of 15.7% over a period of five years to be regarded as control?" It should be pointed out that 10.3% (42 men) of the entire group showed ranges of lead excretion of 300 micrograms and over per liter before incurring lead intoxication. By *our* standards these employees might have been expected to develop symptoms. On the other hand, 5.3% developed plumbism although their urinary lead was below 300 micrograms per liter. Possible explanations are given herewith for some of the cases in this latter classification.

It will be noted that three of these men (0.7% of total) in the 0—99 range (Table III) developed lead intoxication during the period of study.

One of these three had four episodes of lead intoxication prior to the time covered by this report. In the case of the second individual, two samples during the period of study indicated a value in excess of 300 micrograms per liter. The fact that his urinary lead excretion did reach 300 micrograms per liter suggests that he had a peak of absorption at that time, compatible with subsequent intoxication.

The third man had an average excretory value of 75 micrograms of lead per liter for five years. The highest value during this period was 240 micrograms per liter. Lead in the blood at the time of his intoxication was 83 micrograms per 100 grams of whole blood. This suggests the possibility of incorrect diagnosis in view of the findings in most of the other cases. However, the concentration of lead in the blood could be compatible with some degree of intoxication. It should be pointed out that these three men were exposed for an average period of 7.3 years prior to the study, thus making a total average exposure to lead of 12.3 years.

While working conditions in the plants offered a greater hazard previous to the period of the study, these have since been materially improved.

The analysis of spot urine samples will not necessarily provide a reliable index for control if there are gaps in sampling, especially during job transfers, changed manufacturing processes, or altered occupational environments such as may result from a breakdown in the ventilating system.

Of the twelve men in the 100—199 range (Table III) five reached values of urinary lead excretion in excess of 300 micrograms per liter before intoxication developed. These individuals are also affected by the factors mentioned above. Four others had attacks of lead poisoning before the study but here again no records of urinary lead excretion are available. Evidence of impaired renal function was disclosed in the tenth employee of this group. A phenolsulphonephthalein test showed a two-hour dye output of 28%. The average normal value with intramuscular injection is 70%. His urinary lead was low but the amount of lead in the blood was dangerously high. There seems to be little doubt that his lead intoxication was genuine. The impaired efficiency of the kidneys offers a possible explanation for the discrepancy in concentrations of lead in the blood and urine. Two individuals remain in the range of 100 to 199 micrograms of lead per liter of urine, whose intoxication cannot be satisfactorily explained, because of the absence of records.

Two of the seven men in the 200—299 range, gave a history of previous episodes of lead intoxication. No other records are available. The remaining five have no such background and never reached an excretory value as high as 300 micrograms per liter during the study.

We have inferred that if the excretion of lead in the urine is kept below 300 micrograms per liter the frequency of occurrence of lead intoxication will be low. In the whole group of 406 men, 301 showed ranges of excretion of lead below 300 micrograms per liter of urine. Twenty-two of the 301 men developed intoxication but 14 of these might be explained on the basis of past episodes of the disease. Eliminating these "explained" cases therefore, actually only eight individuals or 1.9% developed intoxication where it would not be expected to occur according to our indices of control. In the remaining 105 men showing ranges of lead excretion *above* 300 micrograms per liter of urine, only 42 employee (10.3%) developed intoxication although it might have been expected to occur in *all* of them.

One man showed excretion values ranging from 190 to 1220 micrograms of lead per liter of urine for a period of 18 months without developing the disease. The average of 48 spot samples on this individual was 716 micrograms per liter.

High Levels

EVEN THOUGH there is evidence that some men can maintain very high levels of urinary lead excretion for long periods without symptoms or signs of plumbism, it is possible that such values cannot be maintained indefinitely without eventual intoxication. Table IV shows that 68 of the entire group of 406 men (16.7%) had average concentrations of urinary lead of over 400 micrograms per liter for varying periods of exposure without apparent toxic symptoms.

TABLE IV

MEN SHOWING CONCENTRATIONS OF LEAD IN URINE OVER 400 MICROGRAMS PER LITER WITHOUT INTOXICATION

Number of Men	% of 406 Men	Number of Years Lead Excretion Remained Above 400 Micrograms Per Liter
53	13.0	1
10	2.4	2
4	0.9	3
1	0.2	4
68	16.7	

TABLE V

MEN WITH CONCENTRATIONS OF LEAD IN URINE OVER 400 MICROGRAMS PER LITER DIAGNOSED AS CASES OF LEAD INTOXICATION

Number of Men	Percentage of Entire Group of Men (406)	Number of Years Lead Excretion Remained Above 400 Micrograms Per Liter Before Intoxication Occurred
15	3.7	1
2	0.4	2
17	4.1	

Of the original 64 men diagnosed as cases of lead intoxication, 17 maintained lead excretion values in excess of 400 micrograms per liter for periods of one to two years before intoxication occurred.

Table V gives the distribution of these cases.

In the 64 cases of lead intoxication under study one instance of plumbism occurred at the end of six months. This was the shortest period of exposure before intoxication was observed in any single case. During this six-month period, the average atmospheric concentration of lead was 8.1 milligrams per 10 cubic meters of air, and the average excretion of lead in the urine was 443 micrograms per liter.

Discussion

IN THE 406 cases reported here, one man was diagnosed as a case of lead intoxication although his excretion of urinary lead averaged only 75 micrograms per liter. There is no record that his excretion values were ever otherwise, or that he had any previous episodes of lead intoxication. Thus to make a control guide infallible, at least as demonstrated by these cases, it would be necessary to establish a rather low value in order to include those questionable instances which one finds when dealing with substances in vivo.

Up to the present time, the study indicates that over a period of 12 years, 98% of the men in this group failed to develop lead intoxication as long as absorption remained under a level indicated by an excretory value below 300 micrograms of lead per liter of urine.

In our experience over the past five years, the analysis of spot samples of urine has been justified as a useful index for controlling lead intoxication.

One should recognize that the value of 300 micrograms of lead per liter of urine is not meant to be an unqualified criterion of intoxication. We have adopted the maximal concentration of 300 micrograms of lead per liter of urine as a level beyond which we may anticipate toxic manifestations in a majority of cases, and below which the incidence of lead poisoning will be low.

References

1. HAMLIN, L. E. and WEBER, H. J.: Control of a Lead Hazard in the Brass Foundry Industry. *J. of Ind. Hyg. & Tox.*, 29:6, 1947.

2. WEBER, H. J.: Some Experiences with Polarographic Methods in Controlling a Lead Hazard in Brass Foundries. *J. of Ind. Hyg. & Tox.*, 29:3, May, 1947.

American Conference of Governmental Industrial Hygienists
Eleventh Annual Meeting

American Industrial Hygiene Association Tenth Annual Meeting
April 2-7, 1949, Detroit, Michigan

E. D. SALLEE, American Can Company
C. O. SAPPINGTON, M.D., D.P.H.

MEETINGS of this organization were held on April 2-5. On Saturday, April 2, the Conference meetings were divided into four sections, namely, the chemists, engineers, nurses and physicians. The chemists' meeting, for which DR. R. E. SCHRENK, Industrial Hygiene Division, U.S. Public Health Service, and DR. R. E. MOSER, Industrial Hygiene Section, Oregon Board of Health, served as chairmen, took up such subjects as determination of free silica, the use of x-ray diffraction analysis, methods for analysis of lead-in-air and lead-in-urine, physical methods for analysis of organic solvents, determination of oxides of nitrogen and, of course, touched on atmospheric pollution and on radiation problems. Noticeable by the absence of discussion on the subject were the methods for dust counting.

JOHN C. SOET, Bureau of Industrial Health, Michigan Department of Health and C. D. YAFFEE, Industrial Hygiene Division, U.S. Public Health Service, served as chairmen of the engineering section. Forty-four different questions were discussed, each of which concerned routine engineering problems too detailed to cover here. The Arkansas representative reported on the hazards of carbon monoxide due to space heaters used in tourist cabins. It seems that Arkansas has had a considerable number of fatalities from carbon monoxide in these tourist cabins. Subsequent discussion revealed that this occurrence is common in other states also. DR. PETRIE of Georgia announced that concerted study was being made of carbon monoxide space heaters in his State and that a well organized scientific study was being made.

Although the nurses' section was not attended by the writers, it is understood that this section for which F. RUTH KAHL, Industrial Hygiene Division, U.S. Public Health Service, and VERONICA DONNELLY, Division of Industrial Hygiene and Safety Standards, New York State Department of Labor, were chairmen, emphasized the shortage of nurses, who have proper qualification.

In the physicians' section, with DR. HARRY HEIMANN, Division of Industrial Hygiene, U.S. Public Health Service, and DR. L. W. SPOLYAR, Division of Industrial Hygiene, Indiana State Board of Health, as chairmen, the topics discussed at greatest length were air pollution and a definition of the word "health" with considerable discussion being held on whether the definition of health should include the concept of "well being." Toxicity of insecticides in relation to man was also discussed.

On Monday, April 4, DR. H. K. ABRAMS, Bureau of Adult Health, California Department of Public Health, gave an excellent talk on the health hazards associated with agricultural chemicals including insecticides and pesticides as well as fertilizers. It was pointed out that there are approximately 80 thousand different kinds of insects in the United States, five thousand of which are of importance in agricultural problems. It is estimated that insect damage in the United States is around four billion dollars annually. The discussion was centered around the newer pesticides which have largely replaced older materials such as arsenates and Paris green. Evidence shows that DDT is stored in the body fat and may have a possible cumulative action in man. It was also pointed out that DDT can be excreted in milk and butter fat. DR. ABRAMS cautioned that the less toxic insecticides may not be the safest since more of the less toxic material may be required for effective control.

The resume of these meetings up to the sessions on Thursday, April 7, is part of a report presented to the Chicago Section, AMERICAN INDUSTRIAL HYGIENE ASSOCIATION on May 18, 1949 by MR. SALLEE who served as one of the Local Section Councilors from that Section. The resume of the papers presented at the April 7 sessions was prepared by DR. SAPPINGTON with somewhat greater detail on a number of the papers which are to be published in other journals.

LOUIS F. GARBER, Section of Environmental Sanitation, Department of Public Health and Welfare of Missouri gave an excellent discussion on the lead hazard to workers exposed to galena. In this study exposures to galena expressed as lead in air were frequently over 10 mgs. per 10 cubic meters of air with some exposures as high as 80 mgs. Very thorough clinical examinations were made both in winter and summer on 16 concentrate handlers and it was found that in no case was clinical health damage found.

A reivew of the study of x-ray fluoroscopy in shoe fitting in Detroit was presented by W. G. FREDRICK and R. G. SMITH, both of the Bureau of Industrial Hygiene, Detroit Department of Health. The study included inspection of 200 shoe fitting machines in Detroit and it was found that practically all of these were considered to be potentially unsafe for the shoe clerks. In a few cases machines were found which were considered potentially dangerous to health of customers buying the shoes. It was found that it was possible to adjust or modify practically all of these units so that they would be considered safe and be within the regulations set up by the Detroit Health Department.

Considerable discussion was held on the role of industrial hygiene agencies in air pollution programs due to the fact that most of these agencies do not feel that they have sufficient personnel to satisfactorily take care of such problems. The subject was introduced by H. G. DYKTOR, Division of Air Pollution Control, Cleveland Department of Public Health and Welfare. From past experience it seems that a great deal of the air pollution work has been in connection with the answering of complaints registered by residents in a community. In many cases these may be entirely "nuisance" complaints. At any rate, it is quite apparent that officials and the public alike are becoming considerably more interested in air pollution and that we can expect more action in the not too distant future.

American Industrial Hygiene Association

Tuesday, April 5, Afternoon Session

THE FIRST series of papers was a departure from the normal theme in that they reported experiences of plants in exposures which significantly exceeded the generally recognized tolerances for health hazards and yet no apparent health damage was experienced.

DR. JAMES H. STERNER, Eastman Kodak Company, and President of the American Industrial Hygiene Association, and his associates, gave a paper on a study of butyl alcohol exposures in which a group of employees was exposed to normal butanol vapors at a coating operation over a period of 10 years. Repeated measurements of the exposures showed that the average for the firstr part of the 10-year exposure period was around 200 parts per million. The average for the greater part of the period was around 100 p.p.m. The generally recognized maximal allowable concentration value is now 50 p.p.m. and some authorities have indicated that they would prefer it as low as 25 or 35 p.p.m. DR. STERNER'S report indicated that employees in his company had been exposed to an average of 100 p.p.m. for the entire working shifts with no health damage. It appears that for persons accustomed to butanol vapors, a tolerance level need be no lower than 100 p.p.m.

FREDRICK MALLETTE and WARREN L. HOGUE, JR., both of the Firestone Tire and Rubber Company, and DR. EMMERICH VON HAAN, Ohio State University, reported on x-ray studies of workers exposed to talc in the rubber industry in which a large group of men were exposed to fairly high concentrations of talc dust for periods of 10 years, with a few as high as 30 years. Even though the exposures to talc were of fairly high concentration, x-ray studies showed no change in the workers not consistent with their age.

KARL L. DUNN, Corning Glass Works, reported on the exposure history of the Corning Glass Company in which glass workers were subjected to high intensity infra-red radiation over long periods with no apparent health damage. This is in contrast to the British report wherein high incidence of cataract among a group of British glass workers was reported.

FRANK L. OGLESBY of the Tennessee Eastman Corporation reported on acetone concentrations not associated with injury. It was pointed out that in their experience acetone is detected by the odor upon first

entry into a contaminated area at a concentration of approximately 300 to 400 p.p.m. Continued presence in this concentration level produces fatiguing of the olfactory sense and an inability to detect somewhat higher concentrations. The irritant effect of acetone was reported to be extremely mild, resulting in slight discomfort to the eyes in concentrations of 2000 to 3000 p.p.m. with no irritation after the exposure has ceased. It was MR. OGLESBY'S opinion that concentrations of acetone up to the 1500 to 2000 p.p.m. range would be tolerated without injury or serious objectionable effect for continuous eight-hour exposure and that intermittent exposures totaling not over four hours daily would be permissible in concentrations up to 2500 to 3000 p.p.m.

A preliminary report on environmental aspects of industrial hygiene in the ferrous foundries in Illinois was made by LUCIEN E. RENES, U.S. Public Health Service. He reported that worker exposure by occupation is being studied and also other environmental factors, such as dust particle size measurements, noise and illumination. Complete medical and dental examinations are being used to augment the engineering and chemical phases of the work, all designed for the prevention of occupational diseases in the ferrous foundry industries.

Thursday, April 7, Morning Session

THIS SESSION was a symposium on beryllium, with DR. ROBERT A. KEHOE presiding.

DR. H. E. STOKINGER and associates, Atomic Energy Project, University of Rochester, presented a paper on "Acute Experimental Beryllium Poisoning by Inhalation Exposures." The increasing number of industrial cases of acute beryllium poisoning has made urgent a thorough investigation of beryllium toxicity. In studies that have been made in progress for two years, the toxicity limits of a *soluble* compound, $BeSO_4$, have been established for exposures of from 15 to 100 days at beryllium concentrations of from 5 mg. to 40 mg. per cubic meter, and the histologic character of the response in 11 animal species defined and compared to that in man. The toxic limits of exposure and response in an *insoluble* beryllium compound, BeO, have been defined in a similar group of studies. A striking relation of particle porosity to toxicity of this oxide has been established for various grades obtained from different temperatures of calcining; increased toxicity was correlated with greater particle porosity and lower temperatures of calcining. In the search for criteria of early and mild changes produced by both compounds, changes (a) in the arterial oxygen tension, (b) in certain blood cell lipid fractions, and (c) in serum alkaline phosphatases have shown promise as diagnostic aids. A search for additional factors ("X" factor of Gardner) modifying the response to beryllium has demonstrated (a) that inhaled HF vapor potentiates the toxicity of beryllium in rats, and (b) that exercise markedly increases the mortality in hamsters. Other forms of physiologic stress in the form of pulmonary infection with or without adjuvants have shown mostly negative results: (1) that tuberculosis does not predispose to acute berylliosis in guinea pigs, (2) nor was the reverse chain established in rats, because a gram-negative bacillus isolated from beryllium exposed animals appeared to increase the susceptibility to beryllium in an occasional animal only. Furthermore, (3) oil pneumonitis exhibited some possible influence, but (4) H-pertussus and pneumococci administered intratracheally with or without hyaluronidase had no remarkable influence on the course of beryllium poisoning.

MERRILL EISENBUD and associates, U.S. Atomic Energy Commission, discussed the subject "Non-occupational Berylliosis." Summary of this paper is as follows: Eleven cases of chronic beryllium poisoning (berylliosis) have been reported among residents in the vicinity of a beryllium producing plant. In none of the cases was there a history of occupational exposure to beryllium. Ten of the cases resided within ¾ mile of the plant. The distribution of cases with respect to the plant indicates that the incidence of disease was a function of the concentration to which the residents were exposed. The eleventh case resided almost two miles from the plant but occurred in a member of the household of an employee of this plant. This case is believed to have resulted from atmospheric contamination introduced to the household by work clothes of the employee. Based on air analyses dur-

ing simulated home cleaning of work clothes, a daily laundering can result in the inhalation of 17 micrograms of beryllium. An extensive air analysis program conducted in the vicinity of the plant yielded useful data on the levels of atmospheric contamination. The concentration at ¾ mile is of particular interest because the lower concentrations beyond this distance have not produced known cases in the seven years that this plant has been in active operation. It is estimated that the average concentration ¾ mile from the plant during this period of time ranged from 0.01 to 0.1 milligram per cubic meter. The cases appear to have resulted from exposure prior to 1946, when the ¾ mile concentration is estimated to have been approximately 0.1 milligram per cubic meter. The average daily concentrations of beryllium in the vicinity of a plant producing and processing beryllium should be limited to 0.01 micrograms per cubic meter. There is no justification for applying these data as criteria in judging the hazard of exposed workers in the beryllium industry. The plant in question has had a comparatively low incidence of berylliosis, despite the relatively high exposure to atmospheric contamination within the plant. No satisfactory explanation can be given for this discrepancy, but the specific toxicity of the "neighborhood" contamination may be enhanced by smaller particle size than would be expected within the plant. The minimal exposure of the affected residents is consistent with the low beryllium content of tissues from two cases which have been autopsied. The lungs from these cases contained 0.07 and 0.10 micrograms of beryllium per 100 grams of wet tissue. The air analyses data, when related to the amount of effluent from this plant, are found to be in close agreement with theoretical methods of estimation proposed by Sutton, and Bosenquet and Pearson.

DR. ARTHUR J. VORWALD, The Saranac Laboratories, presented a discussion of "Experimental Production of Lesions Simulating Pulmonary Granulomatosis in Beryllium Workers—Progress Report." The pulmonary changes observed have occurred in a series of rabbits exposed by inhalation to an atmospheric suspension of silicon dioxide dust composed of ultramicroscopic particles. Animals sacrificed after two, four, six, nine and 12 months of exposure revealed a diffuse cellular reaction involving all portions of the lung uniformly. The reaction differs from that observed heretofore following the inhalation of quartz or any preparation of beryllium tested in the Saranac Laboratory. It is strikingly similar to that seen in histologic preparations of lung tissue from human cases of "pulmonary granulomatosis in beryllium workers." In reference to that disease, the significance of these findings remains to be determined.

Thursday, April 7, Afternoon Session

THIS SESSION presented many different types of subjects, with DRS. V. K. ROWE and L. V. GRALLEY presiding.

DR. GORDON C. HARROLD, Industrial Health, Hygiene and Safety Service, Detroit, opened the program with discussion of "Solubility of Lead Salts in Human Pleural Fluid and Blood Serum." The determination of the solubility of lead oxide, lead carbonate, lead chromate and lead titanate in lung fluid and in blood serum is compared with the solubility of lead oxide and carbonate in human lung fluid and blood serum has been found comparable to results obtained with animal serums by other investigators. The solubility of lead chromate and titanate have been found to be of a lower order than found for the oxide and carbonate. The solubility of the purified insoluble lead salts such as chromate and titanate in body fluids will vary from that of impure commercial products due to the much greater solubility in lung fluid and blood serum of the impurities. This fact is complicated by the influence of impurities on solubility due to common ion effects. The solubility of impure lead titanate in triple distilled water is over twice the solubility of purified lead titanate. The solubility of impure lead chromate, on the other hand, is greatly lowered in distilled water because of the soluble chromate ion impurities. The observations represent those conditions which will be encountered when commercial lead compounds are used. The data also reflect the importance of certain lead impurities in insoluble lead salt mixtures. The evaluation of this factor has led to the conclusion that purified lead titanate is less soluble (.287 mg. $PbTiO_3$ per liter of body fluid) than the impure salt. The impurities

in lead chromate are probably in the form of free chromate ion and hence do not affect the solubility in lung fluids of the lead salt except as it acts as a common ion to reduce the solubility as it does in distilled water.

DRS. LESLIE SILVERMAN and S. I. HUSAIN, Harvard School of Public Health, discussed "A Study of Nitrogen Oxide Concentration during Arc Welding," summarizing as follows: The purpose of this study was to observe the kinetics of oxide of nitrogen formation particularly with respect to nitrogen dioxide (NO_2). The effects of iron fume concentrations; air recirculation, with and without filtration of the fume; and the use of bare and coated electrodes at various voltages were investigated. Oxides of nitrogen were determined as nitrites and nitrates. With the aid of a freezing trap technique using liquid nitrogen, nitric oxide (NO) was separated from the other oxides and determined. The data obtained indicate that a considerable amount of nitrogen oxides react with iron fume or are absorbed on it. The possibility that N_2O might be present is also indicated but this gas could not be determined because of limited methods. The results indicate that during welding NO_2 concentrations increase in proportion to number of electrodes used. After welding ceases, NO_2 concentrations remain stable or decrease and do not increase kinetically as theory indicates.

STANLEY C. KYLE, Occupational Health Branch, Tennessee Valley Authority, described "Continuous Carbon Monoxide Recording and Signaling Instruments." To fill TVA's needs for a continuous carbon monoxide recorder, it was found necessary to design and build a new instrument. Specifications for this instrument included the following requirements:

1. Field maintenance must be limited to that which could be performed by a nonskilled worker, and periodic maintenance of a more technical type should not be required oftener than once a month.
2. It must produce a continuous graphic record of concentrations throughout a wide response range.
3. It must be sufficiently sensitive to detect in relatively small increments.
4. Response to changes in concentrations must be rapid.
5. A signal warning system activated by the detection system must be a part of the instrument.
6. Size and weight should not seriously limit transportability.
7. The ability to perform faithfully under adverse operating conditions should be an inherent characteristic of the instrument.

The instrument developed and constructed is rugged and mobile; it is capable of continuously recording atmospheric concentrations of carbon monoxide ranging from 0 to 1000 parts per million with an increment sensitivity of less than 10 parts per million. The response, when sampling through 100 feet of hose, has approximately 30 seconds lag. The instrument may be used in research, survey, or control work and is equipped with a triple zone warning signal and control system. It was found that in some instances the use of the instrument automatically brought under control serious and undesirable conditions simply because the signal system gave warning of the situation and the recording of excessive concentrations prompted tighter operating control.

D. G. HUDSON, Duncan-Hudson Company, East Lansing, demonstrated "The Principle of the Fog Filter." The first installation of the "Fog Filter" was at the City of Lansing Asphalt Plant where fine silica dust from the screen room and from the gravel drying kiln passing through a dry collector of the cyclone type was a community nuisance. A 7 x 7 x 14 foot square cabinet was constructed for the filter. It had a vertical dividing partition in the middle and half way to the bottom, providing a "U"1shaped passage for the dust-laden air. A number of spray nozzles were placed on the inner walls, facing at right angles the path of air travel. After experimentation with various spray nozzles and pump pressures, it was found that at least 300 pounds and preferably 500 pounds pressure was necessary. The wet dusts collected from the filter effluent amounted to about four tons per day and were washed from the filter into a nearby river. It was observed that the use of high pressure fog over a period of three years in this use resulted in an excessive corrosion of the substantial steel structure. For this reason, and because experience indicated a large unit was desir-

able, the steel structure is being replaced by a new unit and is being constructed of wood. Specified materials of construction are more important when fog is used than is the case with conventional air washers.

Since this installation disclosed a high degree of "wetting" ability when high pressure fog was employed, it was believed that a way was presented whereby such fine material could be centrifuged out of the air, since a wetted fine air contaminant would have more mass than when dry. To secure a simulated cyclone action in the wetter gases, advantage was taken of the fact that the kinetics of fog particles from some high pressure nozzles provides an excellent induced draft air pump since all air entrained must necessarily move with substantially the same high velocity as that of the fog particle.

By placing a large number of these nozzles around the inside of a round vertical tower and pointing them in the same angular direction in common horizontal planes, the combined effect of this arrangement was to rotate all of the air mass in the tower about that axis and at a very high angular velocity. Since centrifugal force varies with the square of that velocity, it was now possible to throw to the internal wall of the tower the very fine wetter contaminants. At the internal wall, they become part of the liquid effluent draining to the base where they are washed to any place desired. The "drive" of the high pressure fog also has a scouring action and minimizes the caking of solids on the tower walls. The structure appears quite simple, but to secure maximum efficiency the problem of design for each use is complicated and not unlike the design problems associated with automotive manifolding design. To illustrate the efficiency of this cyclone or "vortex" principle, we have the following figures from an installation at a fertilizer super-phosphate acidulating unit where acid fumes are exhausted to the air:

Gas flow	4,500 c.f.m.
Inlet gas temperature	180 deg. F.
Outlet gas temperature	150 deg. F.
Water used	30 g.p.m.
Inlet solids	4.78 grains plus 14.15 grains/cu. ft. of other material.
Efficiency (weight basis)	90.6 to 93.4% total weight 99.2 to 98.4% fluorides 97.9 to 98.5% SO_4 89.3 to 91.6% H_2SO_4

All of the solids were less than 5 microns in size. Various improvements have since been incorporated in the design which will improve the efficiency. One of the first employments of the high pressure fog principle was a small lead smelter where old automobile battery plates are melted down for lead recovery. This unit is now being replaced with a larger one for increased efficiency, but the results will be of interest. They are as follows:

	Dust count m.p.p.c.f.	Total wgt. in grains/cu. ft.	Lead in grains/ cu. ft.	Organic Material in grains/ cu. ft.
At filter inlet	64,350	9.64	4.17	5.32
At filter outlet	5,936	2.85	0.40	2.16
Collection efficiency	91%	70%	90%	59%

All of the air contaminants at this smelter were 3 microns or less in size. An analysis of air-borne particle sizes before and after filtratio nshows a gain in median particle size of a little over one micron, due no doubt to agglomeration, as well as selection. Other places where this new principle has been employed are in the handling of lime fumes from a new recalcining process of waste water-softening salts at the City of Lansing Water Works and in handling maleic and phthalic fumes at one of the chemical manufacturers. In the first instance, 300 c.f.m. of very dense and high temperature fumes were satisfactorily handled by 10 g.p.m. water, while in the latter, 2000 c.f.m. at normal temperature were handled by less than 25 g.p.m.

The theory of operation is wide open for speculation by theoretical physical chemists. There may be gravitational attraction, random impact, surface potentials and electrodynamics as factors, in addition to those of wetting and centrifuging. An interesting fact about high pressure fog as an air filter medium is that while, as would be expected, water soluble air contaminants are more readily collected than those that are non-water soluble, nevertheless the latter can be removed by this method. As an illustration, wax has been taken from the fumes at the lead smelter mentioned above. In addition, the effluent water is almost black with suspended carbon. This is without recirculation. There also seems to be some relationship between the chemical activity of the

particular contaminant and the degree of facility or degree of high pressure required with which fog will effect its removal.

Generally speaking, the use of high pressure fog will employ 1/3 to 1/2 of the amount of water required in a conventional low pressure scrubbing tower. The effluent has to be disposed of in a porous settling basin if it cannot be disposed of otherwise. In some cases, it can be re-circulated, either for economy of water or to build up a chemical concentration in the liquid for its economic value. If water is re-circulated, it generally must be filtered to prevent the clogging of the small orifice nozzles. Its initial and operation costs lie between the conventional wet dust collector on one hand and the bag house and electrostatic precipitator on the other. Its efficiency will vary with the job for which it has to be designed. It is a safe and simple structure to meet the increasing demands for the control of air pollution. The high pressure fog system is a new tool for both internal and external air pollution problems. It will handle many non-water soluble air contaminants and effect a certain amount of deodorizing. It is a specialty and is not intended for the usual air filter problems where the contaminant particle sizes are more than ten microns.

JACK BALIFF and associates, Division of Industrial Hygiene and Safety Standards, New York State Department of Labor, discussed "Transport Velocities for Industrial Dusts—An Experimental Study" (preliminary report). Actual experimental data on the minimal velocities required for the transport of dusts in industrial exhaust systems has been needed by industrial hygiene engineers for many years. This paper reported the first results of such a study on 30 industrial dusts. Additional tests will be conducted with other dusts. The test set-up consists of a "U"-shaped run of 6 in. diameter pipe having a total length at approximately 50 feet with an initial straight horizontal run of 20 feet with transparent sections to visually observe the transport and deposition of the dust under study. Samples of dusts collected from exhaust systems were slowly fed into one end of the pipe, at controlled decreasing air flows, observing the following critical velocities: (1) beginning to bounce; (2) beginning to roll; (3) beginning to settle; (4) complete settling. Then with increasing air flow, the velocities at which the settled dust began to move and the velocities at which the pipe would become cleaned were also noted. The dusts used were sized by means of a series of eight Tyler screens and the specific gravity was determined. The results of these preliminary tests were shown in a large table. In general, minimal transport velocities were found to be lower than those quoted in the literature, although fair agreement with Alden was found.

DR. LEONARD J. GOLDWATER and DR. MAURICE E. SHILS, Columbia University School of Public Health, presented the subject "The Role of Nutrition in Detoxification Mechanisms."

Detoxification reactions are now looked upon as changes brought about within the body which convert foreign chemicals into substances which can be excreted. Three main types are recognized: (1) oxidations, (2) reductions and (3) synthetic reactions. There is a large number of oxidation and reduction reactions but only nine conjugation processes have been recognized. The fact that nutritional factors may influence the toxicity of industrial chemicals has long been recognized. That one influencing factor is related to detoxications is suggested by results reported in this paper, namely, that pantothenic acid deficiency results in impaired acetylation. It is further shown that the toxicity of TNT and DNT will vary depending on nutritional factors. It was suggested that this approach to the study of industrial toxicology may lead to a better understanding of "individual susceptibility" and may also lead to improved methods of prophylaxis and treatment for occupational poisonings.

DR. CHARLES P. CARPENTER and associates discussed "The Assay, Grading, and Interpretation of Acute Vapor Toxicity Results on 100 Chemical Compounds." The method used for grading the activity of a chemical on the basis of response of albino rats to a 4-hour vapor pressure in a 14-day observation period was described. A list of 96 compounds representative of the various grades was given, together with a justification of the use of this method at the Range Finding Level of toxicity study. Va-

por concentration activity grades were detailed relative to four-hour inhalation of listed ppm killing two, three, or four of six rats within 14 days. For example, the degree of hazard listed as slight, included grades 1 to 5 inclusive and parts per million concentration from 16,000 to 256,000 inclusive. The degree of hazard called moderate included grades 6 to 9 inclusive and concentrations of ppm from 1,000 to 8,000. The degree of hazard called definite included grades 10 to 13 inclusive and ppm from 62 to 500. The degree of hazard called serious included grades 14 to 19 inclusive and ppm from 1 to 32. The range of ppm was also given for each of the concentrations of ppm. The list of substances can be obtained from the authors.

DR. WILLIAM F. ASHE and associates, Kettering Laboratory of Applied Physiology, College of Medicine, University of Cincinnati, discussed "The Behavior of Mercury in the Animal Organism, Following Inhalation of the Vapor of Metallic Mercury at Toxic Levels in Air—A Preliminary Report." The data presented included the concentrations of mercury found in the urine, blood and body tissues of animals exposed for varying lengths of time to controlled atmospheres containing mercury vapor in concentrations of 1 (0.86), 6 and 30 micrograms per liter 1 (0.86), 6 and 30 mgm/cu. m.). Serious tissue damage was caused by exposure to even the lowest of these concentrations of mercury vapor in air in periods of time not exceeding 12 weeks. In general the extent of tissue damage and the tissue mercury content are functions of both the duration of exposure and the atmospheric mercury concentration. Serious doubt is raised as to the safety of the presently accepted maximal allowable concentration for mercury; namely, 0.1 mgm/cu. m. Additional studies are in progress the threefold purpose of which is as follows: (1) To learn more about the metabolism of mercury in the animal organism following absorption when inhaled as the vapor in air. (2) To find some method, based upon the physiological behavior of mercury, by which the magnitude and severity of exposure to mercury can be determined. (3) To establish with certainty a safe maximal allowable concentration for mercury vapor in air for industry.

DR. LEON LEWIS and associates, School of Public Health, University of California, discussed "Experimental Barium Poisoning."

Acute Toxicity in Mice and Rats: Barium chloride was administered orally and intraperitoneally as a 1% or 10% solution. All animals were starved 12 hours prior to use. Oral dosing was done by means of blunt needle and syringe. The rats were females of the Long-Evans strain, weighing between 125 and 150 grams. The mice were of an inbred strain weighing 20 grams plus or minus 2. Death occurred in from 30 to 180 minutes in the animals dying. All surviving animals were observed for a ten day period. The LD_{50} values were obtained by the graphical method of Miller and Tainter using 40 to 60 animals. The values are shown, as follows:

LD_{50} VALUES IN MICE AND RATS

Route of Administration	LD_{50} mg/kg.	Se	Species
I.P.	65.5	3.2	mice
I.P.	48.5	3.1	rats
I.G.	265	1.2	mice
I.G.	463	33	rats

Subacute Toxicity in Rats: Five groups of 20 female rats were placed on diets containing known added quantities of barium chloride. A sixth group was used as a control. The per cent of added barium chloride ranged from 0.1 to 10% of the diet. The mixed food which was dispensed from a special metabolism cup was allowed ad lib. The food intake was determined daily and the rats were weighed at weekly intervals. All animals in the 5% and 10% groups died prior to completion of the experiment, with a marked depression of the growth curve. The time of 50 deaths in the 5% group was six days (range 5-28) and for the 10% group was four days (range 2-8). There were three deaths in the group receiving 1%. All surviving animals were killed on the 35th day. The effect of the added barium salt on the growth curve was shown on a graph. Approximately 25% of the rats on the 5% and 1% diet showed light mottling of the kidney cortex on autopsy. Histologic preparations have not yet been completed. Young growing female rats of the Long-Evans strain were given intragastrically the LD_{80} of barium chloride (10% solution).

Groups of 10 animals each were treated at five different time intervals with each of the three agents tested. The time of antidoting was: 10 minutes prior to administration, simultaneously with administration and at intervals of 10, 20 and 30 minutes post-administration of the LD_{50} of barium chloride. The antidoting agents tested were sodium sulfate (600 mg./kg. as a 10% solution) and BAL (50 mg./kg. as a 7% solution in cottonseed oil). The sodium sulfate was given both intragastrically and intraperitoneally. The magnesium sulfate was administered intraperitoneally while the BAL was given intramuscularly. The sodium sulfate administered intragastrically demonstrated protective effects when administered as long as 30 minutes after the bariu mchloride. This confirms the well established protection of the sulfate ion in barium poisoning. When given intraperitoneally, however, the sulfate showed no significant protective effect. Magnesium sulfate offered a moderate degree of protection, although the statistical significance is not too impressive. BAL given intramuscularly may show a protective effect when given prior to administration of the toxic barium salt. The mechanism of this effect is not explainable at the present time.

EFFECT OF ANTIDOTAL SUBSTANCES ON THE SURVIVAL RATE OF YOUNG RATS ADMINISTERED THE LD_{50} OF $BaCl_2$

Compound	Route	Time of Antidoting in Relation to Administration				
		—10	S	10	20	30
Na_2SO_4	I.G.	8/10	1/10	3/10	0/10	0/10
Na_2SO_4	I.P.	7/10	4/10	8/10	3/10	8/10
$MgSO_4$	I.P.	4/10	3/10	5/10	0/10	6/10
BAL	I.M.	0/10	7/10	3/10	9/10	9/10

This introductory report serves as the first step in the study of barium toxicity preliminary to an investigation of the hazards and treatment of barium poisoning in industry.

A. M. ADAMS and associates, The Dow Chemical Company, presented data on "The Acute and Chronic Vapor Toxicity of 1,1,1-Trichloroethane (methyl chloroform) for Laboratory Animals." Experiments of limited scope revealed that 1,1,1-trichloroethane has a very low acute and chronic vapor toxicity which is similar to that of dichloromethane. The following exposures permitted 50% survival in rats: three hours at 18,000 p.p.m., and seven hours at 14,000 p.p.m. The following exposures permitted 100% survival in rats: 0.3 hours at 18,000 p.p.m., three hours at 10,000 p.p.m., and seven hours at 8,000 p.p.m. The principal toxic effect produced by single exposures was depression of the central nervous system typical of an "anesthetic" agent. Minor fatty changes in the liver were produced by very severe exposures. The results of repeated seven-hour exposures, five days a week, four to 10 weeks, may be summarized as follows: (a) At 5,000 p.p.m., rats and rabbits showed only a slight retardation of growth. Guinea pigs showed moderate fatty changes in the liver, testicular degeneration and poor growth. (b) At 3,000 p.p.m., rats and monkeys suffered no adverse effects, whereas guinea pigs showed only minimal effects consisting of slight liver damage and growth depression. (c) At 1,500 and at 650 p.p.m., guinea pigs exhibited a barely significant retardation of growth but no organic injury. The animal data indicate that the permissible exposures for man will be greater than those for such substances as trichloroethylene and tetrachloroethylene and will be practically as great as those for dichloromethane.

The Use of Beryllium in New York State

JOHN E. SILSON, M.D., Medical Unit
LAWRENCE P. BENJAMIN, CH.E., Engineering Unit
SAMUEL C. WILSON, Engineering Unit

THE INCREASED use of beryllium in industry has brought to light several new pathologial reactions among workers handling it. Since these were first noted, many cases have been reported, and beryllium has now been added to the ever expanding group of serious industrial intoxicants. While the problem has not as yet become a major one in New York State, the Division of Industrial Hygiene and Safety Sandards has been alert to the many hazards created by the new uses to which this metal has been put, and is keeping under surveillance the various industries wherein this material is being introduced.

Uses of Beryllium

BERYLLIUM is a light metal, chemically related to calcium. It is extracted from beryl, a beryllium silicate. Until 1931, it existed largely as a laboratory curiosity, and as an ingredient in gas mantles. Since then, however, many new uses for it have been found. Added to other metals, it imparts many desirable qualities to alloys; pure, it has uses in the field of atomic energy. The oxide is an ideal refractory because of its high melting point and nonreactivity; calcined with zinc oxide and silica, it makes an excellent "phosphor" for luminescent lamps, neon signs and cathode ray tubes. For these reasons, the industry has developed from an output of two tons in 1931 to about 2000 tons in 1947, with a high of over 3000 tons during the war.

Pathological Manifestations

TOXIC responses to beryllium occur primarily in the lungs, upper respiratory tract and skin.

Two types of respiratory disease have been described. One is an acute inflammatory reaction, varying from a mild pharyngitis or bronchitis to a massive pulmonary edema.[1] It occurs principally among workers exposed to the soluble salts in the beryllium refining industry, and usually within a few days of a heavy exposure. Complete recovery is the rule, except for an occasional death from pulmonary edema.

The other respiratory form is a chronic pulmonary granulomatosis,[2] developing after several months to years delay. It occurs primarily among workers handling beryllium phosphors, but has also been seen in those exposed to dusts or fumes of the metal and oxide. Cases have been reported among individuals living in the neighborhood of beryllium processing plants.[7] It is characterized by a nodular fibrosis giving a characteristic x-ray picture, and a diffuse thickening of the alveolar walls resulting in increased resistance to gaseous exchange across this membrane. The outstanding symptoms are fatigue, loss of appetite, weight loss out of proportion to the diminished food intake, and severe intractable cough. Treatment is limited to symptomatic relief, and a large percentage of those surviving remain at least partially incapacitated.

Cases are now being reported where the chronic form has apparently developed without further exposure, several years after recovery from the acute.[7] Conversely, it has occasionally been possible to obtain a history of an illness closely resembling the acute form and antedating the chronic by several months or years.[7] Recently, a subacute type combining features of both the acute and chronic, with an intermediate latent period and duration, has been reported.[6]

In addition to the respiratory disease, several cutaneous reactions have been reported. In the beryllium refining industry, cases of acute dermatosis[1] have occurred from contact with the soluble salts. Crystals embedded under the skin have produced foreign body reactions with ulceration.[3] Extensively proliferating granulomata necessitating wide excision have developed following cuts from glass fragments in which particles of beryllium phosphor have remained.[4] Subcutaneous granulomata have also been observed as a "metastatic" manifestation of advanced pulmonary disease.[4]

Reprinted from *Monthly Review*, Division of Industrial Hygiene & Safety Standards, New York State Department of Labor.

Hazards in New York State

THERE are no major beryllium refineries in New York State, and no plants manufacturing phosphors or fluorescent lamp bulbs at the present time. There are, however, a few industries where some form of beryllium is used, and where a potential hazard exists. These are: (1) the "neon" sign industry, including both the coating of tubing with beryllium phosphors and the manufacture of signs from this tubing; (2) industries handling beryllium alloys, particularly the 2% to 4% beryllium copper, and (3) specialized and experimental uses in the ceramics industry.

"Neon" Sign Manufacture

THIS industry in the beginning used only uncoated glass tubing, and obtained various color effects by varying the gases and the color of the glass. More recently, it has developed the use of tubing coated with fluorescent powder, both to enhance the brilliance of the light and to achieve still more varied colors.

At the present time, beryllium containing phosphors are used for all white effects, and to lighten the colors obtained with other salts such as barium and strontium. As a result, the beryllium content of the phosphors may vary from 0 to about 2½%.

Two types of tubing are processed in the neon sign shops: pre-coated, and plain tubing coated in the shop as required. The former has several advantages from a health standpoint. It eliminates the extra hazard involved in coating the tubing in small batches, which is difficult to control. The phosphors of the pre-coated tubing, which are baked on, are more adherent, and less likely to create a dust hazard in subsequent processing. Pre-coated tubing is usually shipped with stoppers in both ends, which also reduces the dust hazard. In addition to the many sign manufacturing shops, there are also a few plants manufacturing pre-coated tubing in New York State, although a good proportion of it comes from the large fluorescent lamp manufacturers.

The most serious potential hazard in the manufacture of fluorescent tubing lies in the mixing, firing, grinding and screening of the phosphors. These operations were not observed in the plants visited, which purchased their phosphors already prepared. The phosphors are weighed out into rotating ball mills; organic solvents, such as butyl acetate, and nitrocellulose are added; and the mill rotated for sufficient time to reduce the material to small particle size and make a smooth, uniform suspension.

Lengths of clean glass tubing are racked up vertically, and connected to manifolds by rubber tubing at both ends. The suspension is forced up to the top of the tubes through the lower manifold, and then allowed to drain down. The lower ends are then disconnected, and the tubes dried by drawing air through the upper manifold. In some installations, only one rack is equipped for filling, and the tubes, while still dripping, are transferred to another for drying. The tubes are then labeled, baked in an oven which burns out the nitrocellulose, and the ends corked prior to storage or shipment.

The operations of weighing the phosphor and filling the ball mills present a dust hazard and should always be conducted under a hood provided with adequate mechanical exhaust ventilation. Once in suspension, this hazard is eliminated. When the liquid is allowed to drip on the floor and dry, phosphor dust may be created by walking over it. Scrupulous attention to housekeeping is essential, therefore, and any spillage should be mopped up as soon as it occurs. Transfer of dripping tubing should be avoided, and racks so designed that the tubing can dry in place after coating. Dry tubing should be stacked vertically over a grill with a pan underneath to catch the particles dropping out. Control is necessary to remove the dust which may be created at the ends of the tubes rolling out of the baking oven. The entire area where the coating operations are performed should be designed so that it can be readily kept clean, with impervious floors which can be washed at frequent intervals. This area should be isolated from other parts of the plant.

As was previously mentioned, many of the plants manufacturing neon signs coat part or all of their tubing themselves. As a rule, they purchase the coating suspension already mixed, so that they do not have to handle the dry powder. Except for this, and the extra handling involved in baking the tubing, they are exposed to all the hazards of the coating industry enumerated

above. Furthermore, because the amount of production is far smaller, the techniques and equipment tend to be less efficient, with more spillage and careless handling. For these reasons, plus the greater hazard in handling tubing in which the phosphor has not been baked on, it would be preferable for small plants such as these, if possible, not to do any coating with beryllium-containing phosphors.

Neon Sign Fabrication

ADDITIONAL hazards occur in the shaping of the signs. The cutting, splicing, and other handling allows considerable powder to escape from the tubes onto the tables and floor, and there is additional contamination from accidental breakage. Cut-off ends and other scraps of tubing are frequently dropped on the floor, where they are ground underfoot. The expansion of the air in the tubing when it is heated for bending may force fine particles of phosphor into the worker's mouth, if, as is usually done, he keeps a rubber tube connected to the work between his lips. Some workers even insert the ends of the coated tubing into their mouths.

Phosphor dust tends to collect about the storage bins when these are improperly designed. The principles governing tube storage and housekeeping that were outlined for the tube coating plants are equally applicable here. Dry sweeping should be avoided, and cleaning should be done with a vacuum cleaner after raking up the broken glass. Since the dust is inevitably stirred up during cleaning, it should be done after hours, and by a worker equipped with a respirator approved for use with toxic dusts. Ends and broken pieces of tubing should not be allowed to accumulate on the tables and floors, but should be deposited in waste bins. A mouthpiece containing glass wool or cotton to filter out the dust should be used for all glass blowing operations, and the filter changed frequently.

All cuts or other breaks in the skin should be properly treated to insure that no particles of glass or powder can enter or remain in them. Workers should wash their hands and faces thoroughly before eating or quitting work. Under no circumstances should food be eaten or stored in the work areas, and a suitable lunch room should be provided. Workers should be advised not to smoke while working, because of the danger of contaminating the cigarette ends with toxic material.

Beryllium Oxide Ceramics

BECAUSE of its high fusion temperature and other favorable characteristics, beryllium oxide is being used in the manufacture of certain specialized ceramics which have to withstand extremely high temperatures. Most of this work is still in an experimental stage. Since the oxide is believed to be toxic, and is used for this purpose in finely powdered form, extreme care should be employed in handling it. All mixing, molding and other preparation should be done in hoods provided with exhaust ventilation, and the baking kilns should have stacks discharging outside the plant. Any testing of the ceramics where flaking or crumbling may occur should be done under a hood. Meticulous housekeeping should be maintained to prevent accumulation of beryllium oxide dust or mixed clay.

Periodic Medical Examinations

WHERE a possible exposure exists, a periodic medical checkup of the workers is desirable in order to make sure that no cases of poisoning are developing. This should include, as a minimum, questioning the worker for symptoms, such as cough, anorexia and fatigue, a careful check of his body weight for evidence of persistent loss, and a chest x-ray.

Summary

1. Serious pulmonary and skin lesions have developed in workers exposed to beryllium and its alloys and compounds, even in comparatively low concentrations.

2. The principle uses of beryllium in New York State are in the handling of beryllium phosphors in the electric sign industry, and in the processing of alloys, chiefly 2% to 4% beryllium copper.

3. All operations such as heating, melting, and pouring of alloys, where beryllium fumes may be generated, should be adequately controlled by exhaust ventilation.

4. All operations where beryllium metal, alloy, oxide or other compound dusts may be created, such as mixing or other handling of phosphors and clays, and cutting, grinding, polishing and other machining of the

metal and its alloys, should be provided with mechanical exhaust ventilation or other controls.

5. Meticulous housekeeping should be maintained in all workrooms where any form of beryllium might be handled. Dry sweeping should be avoided, and cleaning should be done by wet methods, or after working hours with a vacuum cleaner by a worker provided with a respirator. Spills should be cleaned up promptly, and no beryllium containing materials should be allowed to remain on the floors where they could be ground underfoot into dust.

6. No food should be eaten or stored in any workroom where beryllium is handled, nor should smoking be permitted.

7. Careful attention should be given to all breaks in the skin occurring in workers handling beryllium, particularly phosphor-coated glass, to insure that particles of the compound are not retained subcutaneously. Workers should be instructed to report any skin injuries to the plant medical office promptly.

Periodic medical examinations, including a weight check and a chest x-ray, are advisable on all workers exposed to beryllium metal, alloy, oxide or other compounds, in the form of dusts or fumes.

Bibliography

1. VAN ORDSTRAND, H. S., HUGHES, R., DENARDI, J. M., and CARMODY, M. G.: Beryllium Poisoning. *J.A.M.A.*, 129:1084, (1945).

2. HARDY, H. L., and TABERSHAW, I. R.: Delayed Chemical Pneumonitis in Workers Exposed to Beryllium Compounds. *J. Ind. Hyg. & Toxicol.*, 28:197 (1946).

3. Sixth Saranac Symposium, September 29th to October 3rd, 1947. (Proceedings in course of publication).

4. GRIER, R. S., NASH, P., and FREIMAN, D. G.: Skin Lesions in Persons Exposed to Beryllium Compounds. *J. Ind. Hyg. & Toxicol.*, 30:228 (1948).

5. MACHLE, W., and BEYER, E.: Toxicity and Control of Beryllium Hazards. Paper delivered at 18th Annual Convention of the Greater New York Safety Council, April 14th, 1948.

6. AUB, J. C., and GRIER, R. S.: Acute Pneumonitis in Workers Exposed to Beryllium Oxide and Beryllium Metal. To be published soon in the *J. Ind. Hyg. & Toxicol.*

Personal communication from DR. IRVING S. TABERSHAW.

Robert A. Kehoe Receives Knudsen Award

THE WILLIAM S. KNUDSEN Award for the most outstanding contribution of the year in the field of industrial medicine was received by DR. ROBERT A. KEHOE, professor of industrial medicine at the University of Cincinnati College of Medicine, in Detroit on April 7. The Award, which was established in 1938, was given by the AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS at their annual convention. DR. KEHOE has been a member of the faculty at the University of Cincinnati since 1919 and is now head of the department of preventive medicine and industrial health, and director of the Kettering Laboratory of Applied Physiology and Institute of Industrial Health of the university. He has been active in the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION for many years and served as president in 1945-46. He was cited for his "broad vision in the field of industrial health and especially in sensing the importance of various science" and for the promotion of adequate postgraduate training in industrial medicine.